

CASE REPORT

Capture and fusion beats during atrial fibrillation and ventricular tachycardia

A Nabar, L-M Rodriguez, C Timmermans, K Kattenbeck, H J J Wellens

Abstract

Two patients were presented, and two previously unreported observations were made. Patient 1, a 50 year old man with episodic palpitations and dizziness for 10 years, exhibited initiation of idiopathic ventricular tachycardia (VT) by atrial fibrillation (AF). Patient 2, a 43 year old woman with a structurally normal heart but recurrent palpitations for one year, demonstrated fusion and capture beats during simultaneous VT and AF. An explanation is given as to why the latter phenomenon is rarely observed.

(Heart 2000;84:e1)

Keywords: ventricular tachycardia; atrial fibrillation; atrioventricular nodal conduction

Department of
Cardiology, Academic
Hospital Maastricht,
P Debyelaan 25, 6202
AZ, Post bus 5800,
Maastricht,
Netherlands
A Nabar
L-M Rodriguez
C Timmermans
K Kattenbeck
H J J Wellens

Correspondence to:
Dr Rodriguez
email: LM.Rodriguez@
cardio.azm.nl

Accepted 29 February 2000

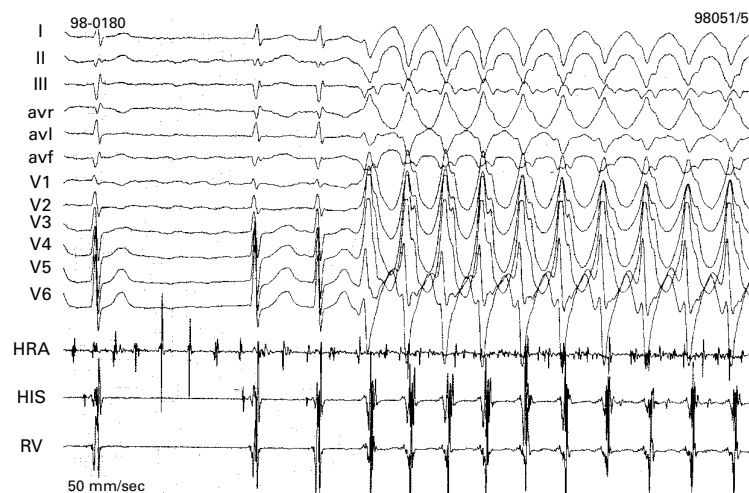


Figure 1 Twelve lead ECG with endocardial recordings from patient 1 during electrophysiological study showing initiation of the ventricular tachycardia (VT) by atrial fibrillation (AF). HRA, high right atrium; HIS, His bundle; RV, right ventricle. Note the long (1140 ms)–short (450 ms) RR cycle length sequence during AF preceding the initiation of VT.

manipulation, sustained atrial fibrillation (AF) occurred, which reinitiated the VT (fig 1). Few narrow QRS beats varying in QRS width and with incomplete right bundle branch morphology were seen, during simultaneous VT and AF, suggestive of fusion beats.

Patient 2 was a 43 year old woman with a structurally normal heart, who had complained of recurrent palpitations for one year, four times associated with collapse. A regular, broad QRS tachycardia (QRS width 130 ms, cycle length 380 ms, and left bundle branch block morphology) with an intermediate axis was documented.

During electrophysiological study, three VTs with left bundle branch block morphology were induced. There was 2:1 and 1:1 ventriculoatrial conduction respectively during the first two VT morphologies. Sustained AF occurred spontaneously and repeatedly initiated the third VT, after a longer RR interval. Narrow QRS beats, resembling sinus QRS complex, were then observed (fig 2).

Discussion

These cases illustrate two uncommon findings, initiation of idiopathic VT during AF and the occurrence of fusion and capture beats following atrioventricular (AV) conduction, during simultaneous VT and AF.

AF provides rapid and irregular RR intervals. Idiopathic left VT is probably based on a re-entrant mechanism, and right ventricular outflow tract tachycardia could be due to triggered activity or abnormal automaticity.¹ In patient 1, a long–short RR interval sequence during AF could create unidirectional block and start re-entry. Long preceding RR intervals have been noted in 77% of cases with “repetitive monomorphic VT and a structurally normal heart”.² In patient 2, irregular RR intervals during AF could lead to rapid escalation of the pause dependent after depolarisations above a critical threshold initiating VT. On the other hand, overdrive excitation during AF could lead to Ca²⁺ overloading and abnormal automaticity.³

Witkamp *et al* showed that in patients with AF, ventricular pacing may prevent atrial impulses from reaching the ventricle.⁴ In patient 1, the absence of ventriculoatrial conduction may indicate lack of retrograde invasion into the conduction system, but the fast VT rate (260 ms) allows only a small window of ventricular excitability. In patient 2, 1:1 and 2:1 ventriculoatrial conduction was present

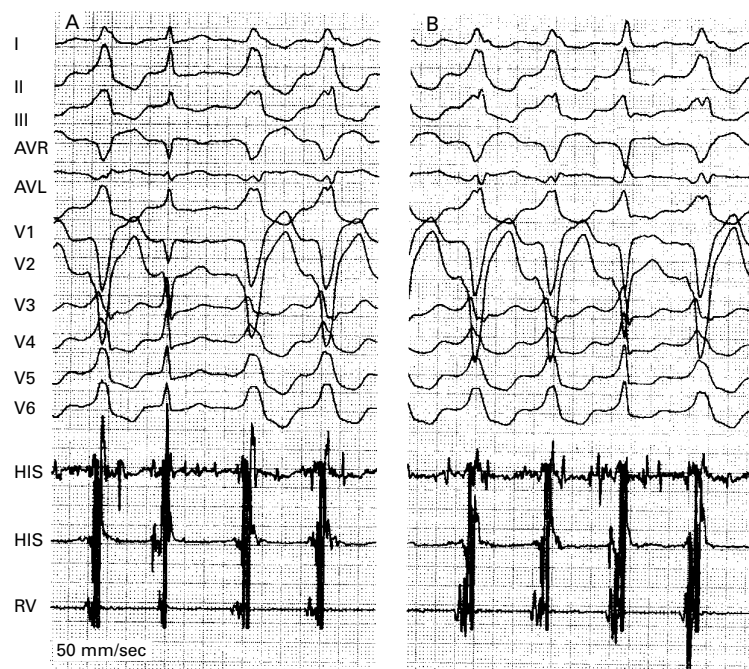


Figure 2 Twelve lead ECG with endocardial recordings from patient 2 during simultaneous VT and AF showing narrow QRS beats. (A) A capture beat; (B) A fusion beat. Note the His bundle electrogram preceding both narrow QRS beats.

during two VT morphologies, suggesting that antegradely conducted supraventricular impulses may capture the ventricle even in patients with a capacity for ventriculoatrial conduction. Apparently, not every beat of the VT penetrated retrogradely in the AV conduction system. Therefore, to have capture and fusion beats during AF and VT, there must be no constant retrograde invasion into the AV conduction system; a sufficiently short antegrade AV nodal refractory period limiting “concealed” AV nodal conduction; and a VT rate allowing the ventricle to be excitable when the supraventricular impulse arrives in the ventricle. This explains why the phenomenon of capture and fusion beats during AF and VT is seen so rarely.

- 1 Wellens HJJ, Rodriguez LM, Smeets JLRM. Ventricular tachycardia in structurally normal hearts. In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology. From cell to bedside*. 2nd ed. Philadelphia: WB Saunders, 1995:780–7.
- 2 Zimmerman M, Maissonblanche P, Cauchemez B, et al. Determinants of the spontaneous ectopic activity in repetitive monomorphic idiopathic ventricular tachycardia. *J Am Coll Cardiol* 1986;7:1219–27.
- 3 Vassalle M. Overdrive suppression and overdrive excitation. In: Rosen MR, Janse MJ, Wit AL, eds. *Cardiac electrophysiology: a textbook*. New York: Futura, 1990:175–90.
- 4 Witkamp FHM, De Jongste NJL, Lie KI, et al. Effect of right ventricular pacing on ventricular rhythm during atrial fibrillation. *J Am Coll Cardiol* 1988;11:539–45.